We claim:

1. A method of treating inflammatory skin disease in a mammal , comprising administering a VEGF antagonist to the mammal, such that the inflammatory skin disease is treated.

2. The method of claim 1, wherein the VEGF antagonist is a fusion polypeptide capable of binding VEGF.

3. The method of claim 2, wherein the fusion polypeptide comprises a VEGF receptor component and a multimerizing component.

4. The method of claim 3, wherein the VEGF receptor component consists essentially of the amino acid sequence of Ig domain 2 of the extracellular domain of a first VEGF receptor and the amino acid sequence of Ig domain 3 of the extracellular domain of a second VEGF receptor.

5. The method of claim 4, wherein the first VEGF receptor is Flt1.

6. The method of claim 4, wherein the second VEGF receptor is Flk1 or Flt4.

7. The method of claim 6, wherein the second VEGF receptor is Flk1 or Flt4.

8. The method of claim 3, wherein the VEGF antagonist is a fusion polypeptide selected from the group consisting of acetylated Flt-1(1-3)-Fc, Flt-1(1-$3_{R->N}$)-Fc, Flt-1(1-$3_{\Delta B}$)-Fc, Flt-1(2-$3_{\Delta B}$)-Fc, Flt-1(2-3)-Fc, Flt-1D2-VEGFR3D3-FcΔC1(a), Flt-1D2-Flk-1D3-FcΔC1(a), and VEGFR1R2-FcΔC1(a).

9. The method of claim 4, wherein Ig domain 2 of the extracellular domain of the first VEGF receptor is upstream or downstreatm of Ig domain 3 of the extracellular domain of the second VEGF receptor.

10. The method of claim 3, wherein the multimerizing component comprises an immunoglobulin domain.

11. The method of claim 10, wherein the immunoglobulin domain is selected from the group consisting of the Fc domain of IgG, the heavy chain of IgG, and the light chain of IgG.

12. The method of claim 10, wherein the immunoglobulin domain is the Fc of IgG1, or a derivative thereof.

13. The method of claim 1, wherein the mammal is a human suffering from an inflammatory skin disease.

14. The method of claim 1, wherein the inflammatory skin disease is psoriasis.

15. The method of claim 14, wherein treatment results in treatment of a symptom associated with psoriasis resulting in reduced severity of a psoriatic lesion, reduced hyperproliferation of keratinocytes, reduced epidermal hyperplasia, reduced reteridges, reversal of epidermal hyperplasia, prevention of infiltration of lymphocytes from dermis into epidermis and/or treatment of parakeratosis and microabcess.

16. The method of claim 1, wherein administration is topical administration, subcutaneous administration, or perhaps intramuscular, intranasal, intrathecal, intraarterial, intravenous, transvaginal, transdermal, or transanal administration.

17. A method of enhancing wound healing in a human comprising administering a VEGF antagonist to the human.

18. The method of claim 17, wherein administration is topical administration.

19. The method of claim 17, wherein administration is subcutaneous administration.

20. The method of claim 17, wherein administration is intramuscular, intranasal, intrathecal, intraarterial, intravenous, transvaginal, transdermal, or transanal administration.